INTERNATIONAL ACADEMIC PUBLICATION DOSSIER

THE VIEN GUT MODEL

Integrated Outpatient Care for Complex Chronic Multimorbidity

Part A – FOUNDATIONAL DOCUMENTS

DOCUMENT A.0

ARCHITECTURAL STATEMENT

From the WHAT–HOW–DATA-to-operate framework to the four verification targets and the dialogue-and-validation roadmap of the Vien Gut Model

Vien Gut Model — International Academic Publication Dossier

First systematic compilation — March 2026

Ho Chi Minh City, Vietnam

AUTHORS & ACADEMIC LEAD

Nguyễn Đình Quang Independent medical researcher | Founder of Vien Gut | Overall designer of the HOW — DATA-to-operate / operational layer

TEAM PARTICIPATING IN THE DESIGN OF HOW AND DATA-TO-OPERATE — VIEN GUT

Nguyễn Đình Quang Huy Participated in the design of HOW — DATA-to-operate | Operations governance, system transfer organization — Vien Gut Model

Huỳnh Phước Đại, Nguyễn Sơn Patient-facing language editing | Communications data management, implementation and transfer support — Vien Gut Model

ACADEMIC SUPPORT & WHAT (GUIDELINE) BENCHMARKING — INTERNATIONAL EXPERT GROUP

Thomas Bardin, Pascal Richette Co-authors of the EULAR Recommendations — together with experts in cardiovascular medicine, nephro-urology, hepatology, diabetes, diagnostic imaging, and biostatistics at Université Paris Cité, France, and Sorbonne University. Transfer of the WHAT from guidelines for the treatment of gout and comorbidities; international benchmarking for WHAT; support for HOW design — Vien Gut Model.

DATA GOVERNANCE TEAM — VIEN GUT

Trương Ánh Dương, Huỳnh Hồng Đức Data governance, transfer support — Vien Gut Model

TREATING PHYSICIANS + MULTIDISCIPLINARY TEAM OF VIEN GUT GENERAL CLINIC

Implementation of clinical HOW — risk stratification, windows of opportunity, longitudinal follow-up, risk control, polypharmacy management, and activation of referral safety valves.

RESEARCH SITES

France–Vietnam Center for Research on Gout and Chronic Diseases

Vien Gut General Clinic — 13A Hồng Hạ, Tân Sơn Hòa, Ho Chi Minh City, Vietnam

POSITION OF THIS DOCUMENT WITHIN THE VIEN GUT MODEL ACADEMIC DOSSIER

Document A.0 is not a document presenting one disease axis in detail, nor is it a document describing a specific operating procedure. A.0 is the architectural statement for the entire Vien Gut Model academic dossier. It defines the central subject to which the dossier is directed, explains why this dossier was created, why four verification targets were chosen as the central frame of reference, and how the entire dossier is organized from academic foundations to dialogue and validation. This positioning has been revised to align with the multilayer architecture

presented clearly in C.1 and to replace the previous structure of A.0, in which Part C could still be misunderstood as both the disease-axis application section and the place where multicenter validation was invited.

To read A.0 in its proper place, it is necessary to understand the four layers of the dossier:

Layer 1 — Basic architecture (Parts A and B): Part A establishes the academic foundation: the central subject of the dossier, the WHAT–HOW–DATA-to-operate framework, the conceptual set, the global HOW gap, and the standardized terminology system. Part B describes the general outpatient operating model: the first clinic visit, the four-phase treatment plan, the conditions for the window of opportunity, the patient's role, and enabling conditions. This layer applies across all disease axes.

Layer 2 — Application of the architecture to each specific disease axis (Part C): Each C document takes one disease axis as the main axis and presents how the A–B architecture is applied to the treatment of that axis together with its related comorbidities. In C.1, gout is the main axis; in subsequent documents, the kidney, cardiovascular, liver, or other disease axes may become the main axis, but all still operate on the same foundational architecture.

Layer 3 — Appendices (protocols and procedures): The appendix set contains specific protocols and procedures serving one or multiple disease axes simultaneously. This is the detailed implementation layer and the point of linkage across the C documents.

Layer 4 — Academic dialogue, evidence benchmarking, and the multicenter validation roadmap (Part D): Part D is where the Vien Gut Model moves from publication to technical dialogue, benchmark comparison with groups that have published corresponding targets, and stepwise progression toward multicenter validation along the roadmap for each target.

READER GUIDE TO A.0

• To understand the WHAT–HOW–DATA-to-operate framework, read A.1.

• To understand the definition of the three architectural layers, read A.2.

• To understand the global HOW gap, read A.3.

• To understand the operational terminology system, read A.4–A.5.

• To understand the general outpatient operating model, read B.1–B.5.

• To understand how this architecture is applied to each disease axis, read C.1–C.n.

• To understand the roadmap for academic dialogue, evidence benchmarking, and multicenter validation, read Part D.

ABSTRACT

Document A.0 presents the architectural statement of the Vien Gut Model academic dossier. The central subject of this dossier is not gout alone, but rather the problem of integrated outpatient care for patients with complex chronic multimorbidity in a context where international guidelines already provide the WHAT but do not yet provide sufficient HOW and DATA-to-operate to coordinate multiple treatment goals in the same patient over time. On that question, the dossier is organized around four verification targets at the target-organ level: crystal-free status in gout, delayed dialysis, reduced heart-failure decompensation, and hepatic recompensation. These are

not entirely new medical targets, but targets for which international evidence of varying strength already indicates attainability in randomized trials, cohort studies, observational studies, or international consensus statements. The Vien Gut Model chooses these four targets as its validation frame of reference because they allow assessment of whether an integrated, individualized, longitudinal outpatient care architecture can translate the WHAT of guidelines into real-world outcomes in patients with complex chronic multimorbidity.

BACKGROUND

Vien Gut began in 2007, at a time when Vietnam was still a low-income country. According to World Bank documentation, Vietnam became a lower-middle-income country in 2009. That context implied real limitations in healthcare resources and a shortage of deeply specialized physicians, while in its early stage Vien Gut operated mainly with a team of general internal medicine doctors.

From its earliest years, Vien Gut was not formed primarily to manage mild gout cases, but to receive patients with late-presenting gout, severe complications, and concurrent complex chronic multimorbidity such as chronic kidney disease, heart failure, cirrhosis, diabetes, secondary adrenal insufficiency, and multiple overlapping pathological spirals. It is precisely in this patient group that the gap between the WHAT of guidelines and the HOW and DATA-to-operate of outpatient practice became most visible, and it is from this point that the Vien Gut Model gradually took shape. This is consistent with the context and source patient population presented in C.1 and aligned with the operating logic of Part B.

Looking back from the present, the most important issue is not how many international guidelines have increased in number, but that clinical practice in patients with severe complicated gout and complex chronic multimorbidity continues to raise the same core question: how to organize outpatient treatment in a way that is sufficiently individualized, sufficiently continuous, and sufficiently safe to pursue multiple high-level treatment targets simultaneously in the same patient. A.0 was written to define clearly how this dossier answers that question and how the entire dossier is organized from academic foundations to dialogue and validation.

OBJECTIVES AND SCOPE OF THE DOCUMENT

Document A.0 has four objectives. First, to define the central subject of the Vien Gut Model academic dossier. Second, to explain why gout is the model's starting axis but not the dossier's only subject. Third, to establish the four verification targets as the central frame of reference for the entire dossier. Fourth, to present the dossier's four-layer architecture, from academic foundations and general operations to disease-axis application, appendices, and the dialogue-and-validation roadmap.

Document A.0 does not provide detailed definitions of the three WHAT–HOW–DATA-to-operate layers, does not describe operating procedures in detail, does not present each disease axis in depth, and does not replace Part D, which is where academic dialogue, evidence benchmarking, and the construction of the multicenter validation roadmap are presented. Those contents belong to A.1–A.5, B.1–B.5, C.1–C.n, and Part D.

1. WHAT IS THE CENTRAL SUBJECT OF THIS DOSSIER?

This dossier is not a dossier solely about gout. Gout is the historical starting point of Vien Gut, the place where the model first accumulated data, clinical experience, and its operational framework. But the true central subject of this dossier is a larger problem: how to deliver integrated outpatient care for patients with complex chronic multimorbidity when international medical literature already provides the WHAT for each disease axis, yet still does not provide sufficient HOW and DATA-to-operate to pursue multiple treatment targets simultaneously in the same patient over time.

International guidelines such as EULAR, ACR, KDIGO, ESC, and EASL [1–5], along with equivalent guidelines, have increasingly improved the WHAT: treatment targets, drug choices, biochemical thresholds, evaluation criteria, and monitoring principles for each disease. However, when multiple guidelines converge on a single patient with four to seven severe diseases at once, what remains missing is not "knowing what to do," but "how to organize doing it," "how to track changes over time," and "on what data to base decisions when guidelines conflict." That gap — the HOW and DATA-to-operate gap — is the true central subject of this dossier.

2. WHY GOUT IS THE MODEL'S STARTING AXIS

Gout is the historical starting axis of the Vien Gut Model and also the axis that allows the most direct and explicit verification by structural imaging among the four verification targets of this dossier. In gout, the urate crystal burden can be observed, quantified, and followed longitudinally by OMERACT ultrasound and/or DECT. This gives the gout axis the role of a methodological point of departure: from a visible structural target, the model gradually expands to the kidney, cardiovascular, and liver axes, where treatment targets are verified mainly through organ function, clinical events, and time-series data.

At the methodological level, gout has a special advantage. Most other chronic diseases are followed through biomarkers or indirect functional indices such as HbA1c, eGFR, EF, or Child–Pugh. In contrast, in gout, the deepest treatment target is the dissolution of the urate crystal burden — a pathological structure that can be directly observed and followed by imaging. For that reason, gout makes it possible to create a closed verification loop between treatment indication, longitudinal monitoring, and confirmation of outcome at the target organ. That is why gout became the most favorable starting axis for building, testing, and standardizing an integrated care model before extending that logic to other disease axes.

However, gout in this dossier is not viewed as an isolated disease. From the original Vien Gut cohort onward, patients with severe complicated gout commonly also had chronic kidney disease, heart failure, cirrhosis, diabetes, metabolic disorders, and complications resulting from prior treatment. Therefore, the role of gout in this dossier is not to narrow the model to a single specialty, but to create a starting axis with the clearest verifiability in the setting of complex chronic multimorbidity.

3. WHY FOUR VERIFICATION TARGETS WERE CHOSEN AS THE CENTRAL FRAME OF REFERENCE

The four verification targets of the Vien Gut Model are not presented as entirely new medical targets. They are four clinical targets for which international literature already provides different levels of evidence showing that they can be achieved: crystal-free status or dissolution of the crystal burden in gout; delaying kidney replacement therapy or preserving kidney function in progressive chronic kidney disease; reducing episodes of worsening heart failure and hospitalization for heart failure; and hepatic recompensation in some patients with decompensated cirrhosis. The Vien Gut Model selects these four targets as verification targets because they have both high clinical value and the capacity to test a larger question: whether an integrated, individualized, longitudinal outpatient care architecture can translate the WHAT of guidelines into practical results in patients with complex chronic multimorbidity.

These four targets are not four targets of gout alone. They are four targets belonging to four independent severe chronic disease axes, coexisting in one patient population and managed simultaneously within one integrated outpatient model. The Vien Gut Model does not claim to have "invented" these targets; its distinctive contribution lies in organizing HOW and DATA-to-operate so that targets already supported by international evidence become more feasible in complex multimorbid outpatient practice.

4. THE FOUR VERIFICATION TARGETS ALREADY HAVE INTERNATIONAL EVIDENCE, BUT REQUIRE AN INDIVIDUALIZED INTEGRATED CARE MODEL TO BE APPLIED IN CLINICAL PRACTICE

The argument block below is used as the direct academic foundation for the section on the four verification targets in Document A.0. The content is preserved from the finalized version.

4.1. The problem at hand

In many severe chronic diseases, international literature no longer stops at the goal of "symptom control," but has moved toward higher and more systemically meaningful treatment targets, such as delaying dialysis initiation, reducing episodes of heart-failure decompensation, achieving hepatic recompensation, and progressively dissolving the urate crystal burden to a very low or no-longer-visible state at the time of assessment in gout. What matters is that these targets are no longer merely theoretical hypotheses: they are supported by a body of international evidence including randomized controlled trials, cohort studies, and systematic reviews. However, the gap between "achievable in research" and "achievable safely and sustainably in outpatient practice" remains large, especially in patients with complex chronic multimorbidity.

4.2. First verification target: delayed dialysis is an evidence-based goal

In advanced chronic kidney disease, the IDEAL trial showed [7] that planned early dialysis initiation did not provide better survival or clearer clinical benefit than later initiation, meaning that the timing of dialysis should not be driven by an isolated eGFR value alone but must be placed in a comprehensive clinical context. Conversely, studies on low-protein diets supplemented with ketoanalogues, from the Garneata trial to recent meta-analyses [8], show that in carefully selected and closely monitored patients, kidney function decline can be slowed and the need for kidney replacement therapy can be postponed. This indicates that "delayed dialysis" is a real target, but one that is feasible only when there is continuous monitoring, nutritional control, early detection of red flags, and timely adjustment across each phase of disease progression.

4.3. Second verification target: reducing heart-failure decompensation is an achievable goal

In heart failure, major studies over recent years have shown fairly clearly that worsening episodes, heart-failure hospitalizations, and recurrent decompensation after an acute episode can be reduced. DAPA-HF showed that dapagliflozin [9] reduced the risk of worsening heart failure or cardiovascular death in HFrEF. EMPEROR-Preserved extended this signal [10] to the preserved-EF group, with a reduction in the composite risk of cardiovascular death or hospitalization for heart failure. AFFIRM-AHF showed [11] that in iron-deficient patients stabilized after an episode of acute heart failure, ferric carboxymaltose reduced heart-failure hospitalizations. Thus, "reducing decompensation" is already a proven target, but medication alone is not enough to keep patients from falling back into the decompensation spiral; an operating system is needed that can stratify risk, provide close longitudinal follow-up after discharge, detect phase transition early, and intervene before full decompensation unfolds.

4.4. Third verification target: hepatic recompensation has been established as an achievable clinical goal

Baveno VII formally laid the foundation [12] for the concept of "hepatic recompensation" through criteria requiring control or removal of the underlying cause, cessation of decompensation manifestations, and sustained improvement in liver function. Subsequent studies across different etiologies have shown that this target can be achieved in a substantial proportion of patients. In HBV-related decompensated cirrhosis, Wang's multicenter study showed [13] that more than half of entecavir-treated patients met Baveno VII recompensation criteria. In

alcohol-related cirrhosis, Hofer showed that sustained abstinence [14] can lead to recompensation and is associated with a clear survival benefit. In HCV-related decompensated cirrhosis, Premkumar's cohort study showed [15] that a significant proportion of patients achieved recompensation after DAA therapy, although the risk of new events remained. This body of evidence shows that "recompensation" is no longer an abstract concept but a practical target that can be pursued, though it becomes real only when the cause is controlled, decompensation episodes are blocked early, and outpatient follow-up is disciplined enough.

4.5. Fourth verification target: in gout, a near-"crystal-free" state or no visible crystal burden at the time of assessment has increasingly clear scientific support

In gout, international literature has not yet fully standardized a single term for "crystal-free" as a universal endpoint, but the chain of imaging and clinical evidence is converging clearly in that direction. Perez-Ruiz showed early on [16] that when urate is lowered sufficiently, tophi shrink faster, meaning that the speed of crystal burden dissolution depends on the degree of urate control. Thiele showed that crystal deposition signs [17] on the surface of hyaline cartilage can disappear completely on ultrasound once sustained normouricemia is achieved. The 5-year NOR-Gout data showed [18,20] that a treat-to-target strategy reduces crystal deposition and dissolves both double contour and tophi in most patients. GOUT-DECTUS 2025 further showed [19] that the crystal core of tophi on DECT can dissolve completely after a prolonged treat-to-target strategy, even though the sonographic morphology of the tophus mass may disappear more slowly. Thus, "crystal-free at the time of assessment" is a target with an increasingly strong scientific foundation, but reaching it in severe gout cases with multiple comorbid conditions requires a model that controls uric acid while also managing flares, polypharmacy, organ injury, and adherence over many years.

4.6. The common meaning of the four verification targets

When these four targets are placed side by side, one very important common point becomes clear: international studies have demonstrated that higher treatment targets are feasible, but they are usually achieved in contexts with selected patients, clear protocols, defined monitoring criteria, sufficiently dense assessment frequency, and tightly controlled adverse-event management teams. From this, a practical inference can be drawn: the issue today is not only "knowing WHAT to do," but having a HOW that is strong enough to bring those targets into real clinical life. In complex chronic multimorbid outpatients, if there is no integrated operational layer, no longitudinal follow-up, no risk stratification, no mechanism to resolve conflicts between multiple treatment goals, and no referral "safety valve" when thresholds are exceeded, then even targets already proven in the literature are very difficult to turn into sustainable real-world outcomes. This is an inferential conclusion drawn from the structure and implementation conditions of the existing evidence.

4.7. Implications for the Vien Gut Model

It is precisely here that the Vien Gut Model approach becomes meaningful: it does not stop at benchmarking guidelines to identify treatment goals, but aims to build an integrated outpatient care model for patients with complex chronic multimorbidity, in which each patient is individualized according to disease stage, multi-organ burden, decompensation risk, treatment tolerance, adherence capacity, and early warning signals. In that logic, delaying dialysis, reducing heart-failure decompensation, achieving hepatic recompensation, and reaching a crystal-free state at the time of assessment in gout are not four disconnected goals, but four "verification targets" for the same operating philosophy: to achieve the high targets of modern medicine in outpatient practice, an integrated model is needed that can simultaneously manage treatment goals, treatment measures, treatment risks, and long-term disease course in each specific patient. This is also the direction the Vien Gut Model is pursuing in order to translate international evidence into verifiable real-world effectiveness.

5. VERIFICATION TARGETS AND ENABLING CONDITIONS ARE TWO DIFFERENT LAYERS

Not every disease present in the Vien Gut cohort is defined as an independent verification target. Clinical practice shows that the model naturally separates into two layers. The first layer consists of the four verification targets — axes with specific target-organ injury that can be followed by imaging, function, or standardized events. The second layer consists of enabling conditions — coexisting diseases or conditions that must be controlled in order for the four primary targets to retain an open window of opportunity.

Diabetes, hypertension, lipid disorders, secondary adrenal insufficiency, chronic anemia, malnutrition, and similar conditions may have major clinical significance, but in this publication dossier they are not designated as independent verification targets. They are managed as mandatory operating conditions to protect the safety margin, keep the window of opportunity open, reduce conflict among disease axes, and preserve the attainability of the four verification targets in outpatient practice. This framework is fully consistent with the role of B.5 across the entire dossier.

6. WHY THE MODEL WAS BUILT IN THE OUTPATIENT SETTING

The HOW gap exists in every treatment environment, but in inpatient care it is often obscured by layers of concentrated resources such as on-site multidisciplinary consultations, continuous monitoring, on-duty nursing, and 24/7 emergency response. When the patient leaves the hospital, those obscuring layers disappear; it is at that moment that the HOW gap becomes fully visible. That is why this model was formed from outpatient practice, rather than as merely a “downscaled” version of a hospital model.

Outpatient care forces HOW to be designed explicitly from the outset: who decides what, when, based on what data, within what SLA, when monitoring intensity is escalated, and when the referral safety valve is activated. Precisely because outpatient care does not have the obscuring resources of inpatient care, an integrated outpatient model can be systematized into documents, standardized into procedures, and progressively transferred to similar settings. This is especially important in LMIC contexts, where resources remain limited but the burden of patients with complex chronic multimorbidity is very high.

Precisely because it was formed from outpatient practice in an LMIC context, this model aims for comparability, standardization, and validation at other centers with similar resource conditions, rather than existing only as a local experience. This is a point that must be maintained consistently from A.0 through Part D.

7. THE FOUR-LAYER ARCHITECTURE OF THE DOSSIER

From the analyses above, the Vien Gut Model academic dossier is organized according to a four-layer architecture.

Layer 1 is the basic architecture, comprising Parts A and B. This is where the academic foundation and the general operating model are established. Without this layer, no C or D document can be properly understood.

Layer 2 consists of documents applying the general architecture to each specific disease axis, namely Part C. Each C document selects one disease axis as the main axis in order to present in depth how the A–B architecture is applied to the treatment of that axis together with its related comorbidities. C.1 is the first document in this layer.

Layer 3 is the appendix set. This is where detailed protocols and procedures are placed, serving one axis or multiple axes simultaneously. This layer reflects the integrated nature of the model: a treatment decision on one axis is often influenced by other axes.

Layer 4 is Part D. This is where the Vien Gut Model moves from publication to academic dialogue, benchmark comparison with groups that have published corresponding targets, and stepwise progression toward multicenter validation along the roadmap for each target. Verification targets are not the same as an invitation to validate; the invitation to validate is the next step and belongs to Layer D, not to Layer C itself.

8. THE SPIRIT OF THIS DOSSIER

8.1. Systematizing a clinical practice journey

This dossier does not claim to have found the final answer to complex chronic multimorbid outpatient care. It is the academic systematization of an integrated clinical practice journey spanning nearly two decades, built in a spirit of full respect for the WHAT of international guidelines while honestly acknowledging the HOW and DATA-to-operate gaps that practice has been compelled to confront. The strength of the dossier does not lie in rejecting the existing literature, but in reorganizing the clinical problem within a more integrated frame of reference.

8.2. Academic dialogue and validation

The four verification targets in this dossier are not a unilateral assertion of the model's ultimate status. They are a frame of reference for academic dialogue, benchmarking against international literature, and stepwise entry into the process of multicenter validation. In that spirit, the model must be confident enough to publish, yet humble enough to place itself within the next phase of critique and validation.

8.3. Systemic risks of relying on AI in complex multimorbidity

In a context where no integrated outpatient care model for complex chronic multimorbidity yet exists, both physicians and patients may hope to rely on AI to fill the gap. However, AI currently creates three serious systemic risks. This is why the Vien Gut Model states clearly that support software must be designed within the WHAT–HOW–DATA-to-operate architecture, not as an external AI substitute for that architecture.

No.	Systemic risk	Clinical consequence
1	AI creates the illusion of having HOW	Advice may sound logical but sits within no operating architecture — there is no responsible actor, no SLA, no safety valve, and no audit trail. It is WHAT mixed with a false HOW.
2	AI lacks DATA-to-operate	It knows only a single snapshot at the moment of questioning, without longitudinal data sequences (18-month eGFR, BNP trend, tophi over time). Decisions are made on snapshots — precisely what the Vien Gut Model has shown to be insufficient.
3	AI blurs clinical accountability	When AI says "the colchicine dose should be reduced because eGFR is low," who is responsible if the patient reduces the dose on their own and a flare erupts? The boundary of responsibility disappears between AI, physician, and patient.

8.4. Integrated WHAT–HOW–DATA-to-operate software is being finalized

One important fact must be stated clearly: at the time this dossier is published (March 2026), the Vien Gut Model has already demonstrated operational capability through human capacity and manually accumulated procedures over nearly two decades — 155 crystal-free patients, case DTH, case LAU. The integrated WHAT–HOW–DATA-to-operate software is in the finalization stage, expected by the end of 2026. This is both a limitation (the model cannot yet be widely transferred before digitization) and evidence (the operating architecture has already been validated in real practice before digitization). The software will be the condition that enables the model to scale and undergo multicenter validation.

9. LIMITS OF THE DOCUMENT'S SCOPE

Document A.0 includes: defining the central subject of the dossier; explaining why gout is the model's starting axis; presenting the four verification targets and distinguishing them from enabling conditions; explaining why the model was built in the outpatient setting; and defining the four-layer architecture of the entire dossier. This is the scope of an architectural statement document, not that of a procedures document or a disease-axis application document.

Document A.0 does not include: detailed definitions of the three WHAT–HOW–DATA-to-operate layers; systematic evidence on the global HOW gap; the concrete operating model; disease-axis application documents; or the detailed dialogue-and-validation roadmap for each target. These contents are presented respectively in A.1–A.5, B.1–B.5, C.1–C.n, and Part D.

10. THE PLACE OF A.0 WITHIN THE VIEN GUT DOCUMENT SYSTEM

A.0 is the entry point to the entire dossier. Readers come to A.0 to understand what question this dossier is answering, why this model was built from outpatient practice, why four verification targets were chosen as the central frame of reference, and how the entire dossier is organized according to a four-layer architecture. After A.0, readers can move into A.1–A.5 to grasp the academic foundation, B.1–B.5 to understand operations, C.1–C.n to read by disease axis, and Part D to follow the roadmap for academic dialogue and multicenter validation.

REFERENCES

[1] FitzGerald JD, et al. 2020 American College of Rheumatology Guideline for the Management of Gout.

[2] Richette P, et al. 2016 updated EULAR evidence-based recommendations for the management of gout.

[3] KDIGO. 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease.

[4] ESC/ACC-AHA guideline documents on heart failure and decompensation prevention.

[5] EASL and related consensus documents on cirrhosis decompensation and recompensation.

[6] NICE NG56 and key international documents on multimorbidity and integrated care.

[7] Cooper BA, Branley P, Bulfone L, et al. A randomized, controlled trial of early versus late initiation of dialysis. N Engl J Med. 2010.

[8] Garneata L, Stancu A, Dragomir D, et al. Effect of low-protein diet supplemented with keto acids on progression of chronic kidney disease. J Ren Nutr. 2013.

[9] McMurray JJV, et al. Dapagliflozin in patients with heart failure and reduced ejection fraction. N Engl J Med. 2019.

[10] Anker SD, et al. Empagliflozin in heart failure with a preserved ejection fraction. N Engl J Med. 2021.

[11] Ponikowski P, et al. Ferric carboxymaltose for iron deficiency at discharge after acute heart failure. Lancet. 2020.

[12] de Franchis R, et al. Baveno VII – Renewing consensus in portal hypertension. J Hepatol. 2022.

[13] Wang Q, et al. Validation of Baveno VII criteria for recompensation in entecavir-treated patients with hepatitis B-related decompensated cirrhosis. J Hepatol. 2022.

[14] Hofer BS, et al. Hepatic recompensation according to Baveno VII criteria is linked to a significant survival benefit in decompensated alcohol-related cirrhosis. Liver Int. 2023.

[15] Premkumar M, et al. Recompensation of chronic hepatitis C-related decompensated cirrhosis following direct-acting antiviral therapy. Gastroenterology. 2024.

[16] Perez-Ruiz F, et al. Effect of urate-lowering therapy on the velocity of size reduction of tophi in chronic gout. Arthritis Rheum. 2002.

[17] Thiele RG, Schlesinger N. Ultrasonography shows disappearance of monosodium urate crystal deposition on hyaline cartilage after sustained normouricemia is achieved. Rheumatol Int. 2010.

[18] Hammer HB, et al. Ultrasound-detected crystal depositions and clinical flares after five years of treat-to-target urate-lowering therapy in gout. 2025.

[19] Pascart T, et al. Time-course of tophus resolution on dual-energy CT and ultrasound after 24 months of a treat-to-target strategy: results from GOUT-DECTUS study. Joint Bone Spine. 2025.

[20] Uhlig T, et al. Remission in gout is possible: 5-year follow-up in the NOR-Gout study. 2025.

Foundational and operational documents within the Vien Gut Model academic dossier: A.1–A.5, B.1–B.5, C.1–C.n.